

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Attorney Docket No.: **ISPH-0787**

Inventors: **Karras and Dobie**

Serial No.: **Not Yet Assigned**

Filing Date: **Herewith**

Examiner: **Not Yet Assigned**

Group Art Unit: **Not Yet Assigned**

Title: **Antisense Modulation of MYD88 Expression**

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By *Jane Massey Licata*
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Sir:

INFORMATION DISCLOSURE STATEMENT

Pursuant to 37 C.F.R. §1.56 and in accordance with 37 C.F.R. §§1.97-1.98, information relating to the above-identified application is hereby disclosed. Inclusion of information in this statement is not to be construed as an admission that this information is material as that term is defined in 37 C.F.R. §1.56(b).

(XX) In accordance with §1.97(b), since this Information Disclosure Statement is being filed either within three months of the filing date of the above-identified application, within three months of the date of entry into the national stage of the above identified application as set forth in §1.491, or before the mailing date of a first Office Action on the merits of the above-identified application, no additional fee is required.

() In accordance with §1.97(c), this Information Disclosure Statement is being filed after the period set forth in §1.97(b) above but before the mailing date of either a Final Action under §1.113 or a Notice of Allowance under §1.311, therefore:

() Certification in Accordance with §1.97(e) is set forth below; or

() The fee of \$180.00 as set forth in §1.17(p) is attached.

() In accordance with §1.97(d), this Information Disclosure Statement is being filed after the mailing date of either a Final Action under §1.113 or a Notice of Allowance under §1.311 but before the payment of the Issue Fee, therefore included are: Certification in Accordance with §1.97(e); Petition Requesting Consideration of the Information Disclosure Statement; and the fee of \$130.00 as set forth in §1.17(I)(1).

() Copies of each of the references listed on the attached Form PTO-1449 (modified) are enclosed herewith.


(XX) In accordance with §1.98(d), copies of some or all of the references listed on the attached Form PTO-1449 (modified) are not enclosed herewith because they were previously submitted to the U.S. Patent and Trademark Office in prior application Serial No. 10/021,707, filed November 23, 2001 for which a claim for priority under 35 U.S.C. §120 has been made in the instant application.

Please charge any deficiency or credit any overpayment to Deposit Account No. 50-1619. This form is submitted in duplicate.

() The relevance of the listed references in a foreign language is as stated in the specification at pages @@.

(XX) All listed references are in the English language.

Respectfully submitted,


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Date: September 26, 2003

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Form PTO-1449 Modified		Docket No. ISPH-0787	Serial No. not yet assigned
List of Patents and Publications Cited by Application (Use several sheets if necessary)		Applicant James Karras et al.	
		Filing Date herewith	Group
U.S. Department of Commerce Patent and Trademark Office			
OTHER DOCUMENTS (Including Author, Title, Date, Pertinent Pages, Etc.)			
	AA	Adachi et al., Targeted disruption of the MyD88 gene results in loss of IL-1- and IL-18-mediated function, <i>Immunity</i> , 1998, 9:143-150	
	AB	Akira et al., The role of Toll-like receptors and MyD88 in innate immune responses, <i>J. Endotoxin Res.</i> , 2000, 6:383-387	
	AC	Akira et al., Toll-like receptors: critical proteins linking innate and acquired immunity, <i>Nat. Immunol.</i> , 2001, 2:675-680	
	AD	Bonnert et al., The cloning and characterization of human MyD88: a member of an IL-1 receptor related family, <i>FEBS Lett.</i> , 1997, 402:81-84	
	AE	Burns et al., MyD88, an adapter protein involved in interleukin-1 signaling, <i>J. Biol. Chem.</i> , 1998, 273:12203-12209	
	AF	Dupraz et al., Dominant negative MyD88 proteins inhibit interleukin-1beta /interferon-gamma -mediated induction of nuclear factor kappa B-dependent nitrite production and apoptosis in beta cells, <i>J. Biol. Chem.</i> , 2000, 275:37672-37678	
	AG	Hardiman et al., Molecular characterization and modular analysis of human MyD88, <i>Oncogene</i> , 1996, 13:2467-2475	
	AH	Lord et al., Complexity of the immediate early response of myeloid cells to terminal differentiation and growth arrest includes ICAM-1, Jun-B and histone variants, <i>Oncogene</i> , 1990, 5:387-396	
	AI	Medzhitov et al., MyD88 is an adaptor protein in the hToll/IL-1 receptor family signaling pathways, <i>Mol. Cell</i> , 1998, 2:253-258	
	AJ	Muzio et al., IRAK (Pelle) family member IRAK-2 and MyD88 as proximal mediators of IL-1 signaling, <i>Science</i> , 1997, 278:1612-1615	
EXAMINER		DATE CONSIDERED	

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U.S. Department of Commerce Patent and Trademark Office			
OTHER DOCUMENTS (Including Author, Title, Date, Pertinent Pages, Etc.)			
	AK	Schmidt et al., Lipopolysaccharide-induced activation of beta2-integrin function in macrophages requires Irak kinase activity, p38 mitogen- activated protein kinase, and the Rap1 GTPase, Mol. Cell. Biol., 2001, 21:438-448	
	AL	Schnare et al., Recognition of CpG DNA is mediated by signaling pathways dependent on the adaptor protein MyD88, Curr. Biol., 2000, 10:1139-1142	
	AM	Takeuchi et al., Toll-like receptors; their physiological role and signal transduction system, Int. Immunopharmacol., 2001, 1:625-635	
	AN	Takeuchi et al., Cutting edge: TLR2-deficient and MyD88-deficient mice are highly susceptible to Staphylococcus aureus infection, J. Immunol., 2000, 165:5392-5396	
	AO	Wang et al., Micrococci and peptidoglycan activate TLR2-->MyD88-->IRAK-->TRAF-->NIK-->IKK-->NF-kappaB signal transduction pathway that induces transcription of interleukin-8, Infect. Immun., 2001, 69:2270-2276	
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